CASE REPORT

Risperidone-induced Premature Ventricular Complexes in Psychosis: A Case Report

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Received on: 06 February 2022; *Accepted on:* 01 March 2023; *Published on:* 14 June 2023

Abstract

Atypical antipsychotics are known to cause cardiovascular side effects, such as prolonging the QTc interval due to blockade of the potassium repolarization channels, and hence they predispose to ventricular arrhythmias, like premature ventricular complexes (PVCs).

Here, we report a case of a male schizophrenic patient who is complaining of palpitations and feeling clinically as well as symptomatically troublesome PVCs associated with risperidone. We consider that PVCs may have been generated by risperidone, with his stressful mental condition, and subsided after withholding it.

Keywords: Antipsychotic agents, Arrhythmia, Cardiac, Risperidone.

Indian Journal of Private Psychiatry (2023): 10.5005/jp-journals-10067-0116

Background

Premature ventricular complexes (PVCs) are irregular, extra heart rhythms, such as ectopic beats caused by early myocardial depolarization. PVCs are always associated with heart disease and other related non-cardiac causes, like disturbance of chemical conditions in the body due to alcohol, illicit drugs, and certain type of medications. Even though it is benign, in recent studies a rate of PVC load more than 24% is found, and it leads to cardiomyopathy and heart failure. In some studies, it has been shown that PVCs are seen in the general population: around 4% on 12-lead electrocardiography (ECG) and 40–75% of patients on 24–48 hour Holter electrocardiogram monitoring.[1] According to the literature, cardiovascular side effects, like deviation of blood pressure and arrhythmias, are side effects of antipsychotics.[2] In other conditions, like congestive heart failure, myocarditis with increased cardiac mortality is rarely seen.[3] Research suggests that PVCs have been reported with antipsychotics, such as iloperidone,[4] aripiprazole,[5] risperidone,[6] thioridazine,[7] and quetiapine.[8]

Case Description

A 40-year-old Saudi male patient separated from his second wife was admitted to our hospital. He presented with deterioration of personal, family, social, and work functions, such as social isolation and financially inappropriate decisions for about 1 year. He also showed disorganized behavior with negative features, such as self-neglect and social withdrawal. He did not know what he was doing and where he was present; he interacted in a strange manner and had poor sleep. He had a history of psychosis 9 years back, with symptoms such as suspiciousness, fearfulness that someone followed him, poor sleep, and lack of concentration. He was using antipsychotics olanzapine, paliperidone, and quetiapine on and off. His history included alcohol abuse but now abstaining, but he used to smoke cigarettes excessively. He was taken to a faith healer, improved, but never came back to baseline, and at the time of presentation to our hospital, he had more negative attitude and disorganized behavior. He had no history of mood disorder, no family history of psychosis, and no history of medical and surgical intervention.

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How to cite this article: Siddiqui JA, Qureshi SF, Shawosh YBA. Risperidone-induced Premature Ventricular Complexes in Psychosis: A Case Report. Ind J Priv Psychiatry 2023;17(2):91–94.

Source of support: Nil

Conflict of interest: Dr Javed Ather Siddiqui is associated as the International Advisory Board member of this journal and this manuscript was subjected to this journal's standard review procedures, with this peer review handled independently of this editorial board member and his research group.

Patient consent statement: The author(s) have obtained written informed consent from the patient for publication of the case report details and related images.

The patient was admitted with a diagnosis of schizophrenia and was started on risperidone 3 mg/day, 1 mg in the morning and 2 mg in the night. He reported strange feeling and chest tightness, palpitation, breathlessness, and fatigue with even rest. We referred him to a physician who advised electrocardiogram and routine blood findings, like complete blood cell count, serum electrolytes, renal liver function tests, prolactin levels, and thyroid function test, which were all within the normal range. Physical examination was normal. Therefore, the chances of physical illnesses were excluded. Electrocardiogram was done and showed multiple PVCs (Figs 1 and 2). The physician advised stopping antipsychotics and starting amlodipine 50 mg once daily and referred him to a cardiologist.

The cardiologist attended the patient, advised him to wear a Holter electrocardiogram monitor for 24–48 hours, and asked him to continue amlodipine 50 mg once daily. After 24 hours, Holter electrocardiogram monitoring showed no significant arrhythmia (Fig. 3). However, the average pulse rate was 60 beats

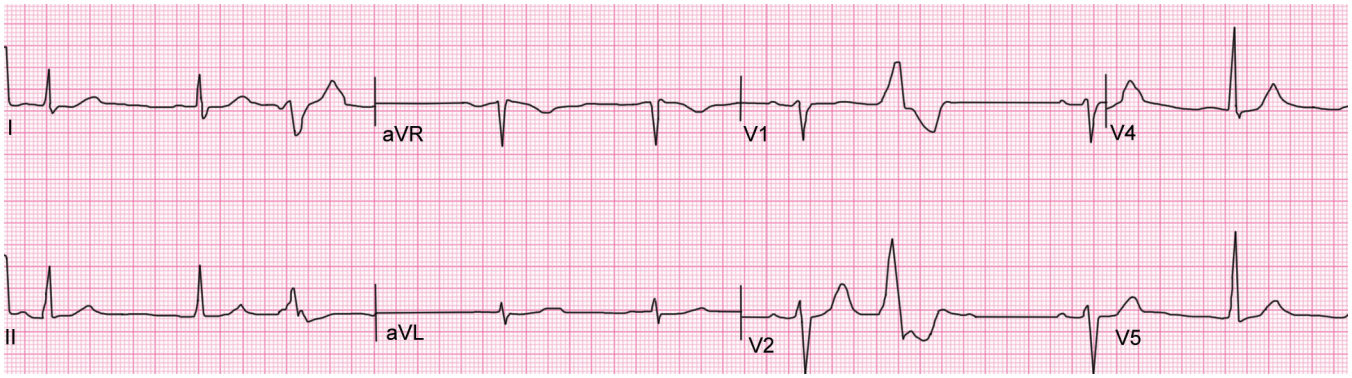

Fig. 1: PVC before stopping risperidone

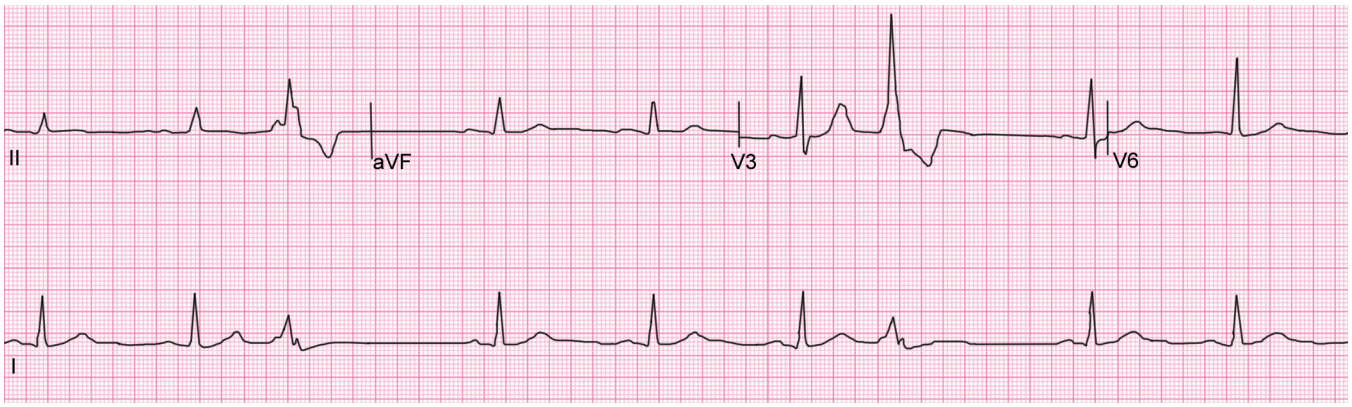

Fig. 2: Frequent PVCs before stopping risperidone

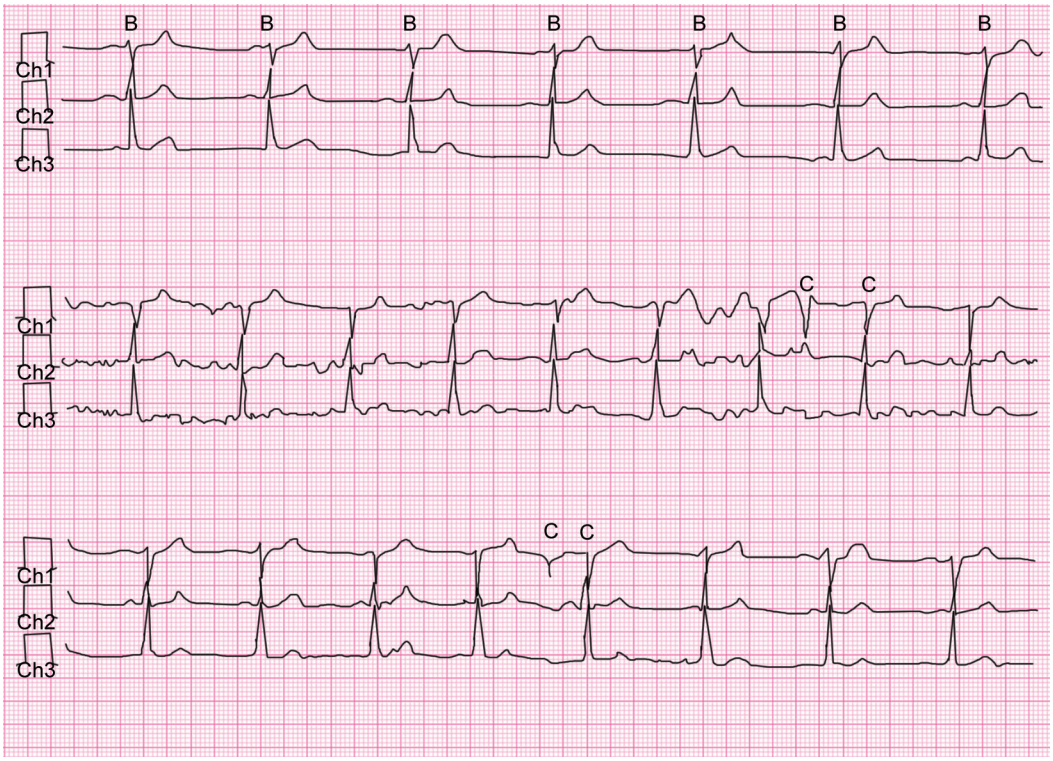

Fig. 3: Holter electrocardiogram monitoring for 24 hours showing no significant arrhythmia

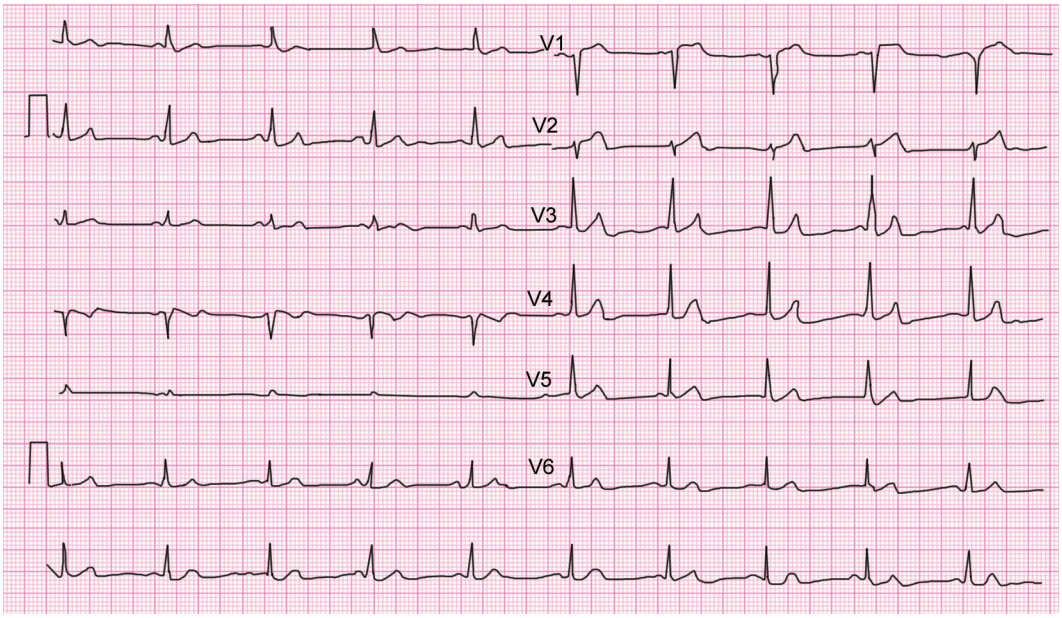

Fig. 4: Electrocardiogram showing normal features after stopping risperidone

per minute with QTc prolongation of 450 ms. On the third day, electrocardiogram was done again, and it was normal (Fig. 4). Naranjo adverse drug reaction (ADR) probability test was done, and the score was 7. It suggested that PVCs were due to risperidone. We referred the patient to a psychologist for counseling, such as life-style changes, avoiding alcohol, and smoking. He was given aripiprazole 10 mg once daily instead of risperidone for his negative features and psychosis. After discontinuation of risperidone, within 2–3 days, he was feeling better, with no more palpitations, chest tightness, fatigue, and breathlessness. The patient remained well settled on 10 mg aripiprazole.

Discussion

Risperidone is a selective monoaminergic antagonist of serotonin and dopamine receptors. It is commonly used for the management of psychosis, and it has unknown affinity for cholinergic muscarinic or β1 and β2 adrenergic receptors. It causes brady-arrhythmia due to the strong temporal association between them. Risperidone therapy and the Naranjo ADR probability rate score was 7, clarifying that PVCs are due to risperidone.

Here, we describe the dramatic finding of sinus bradycardia associated with frequent PVCs, after taking an initially low starting dose of risperidone. The patient had a normal sinus rhythm before the medications, but after starting low doses of risperidone, he had bradycardia and PVCs, which subsided later when risperidone was withheld. Hence, risperidone was one of the causes of such type of arrhythmia. In a study, PVCs developed after a high dose of risperidone, but this patient had symptomatic bradycardia. This condition is seen in young men associated with alcoholic withdrawal. Such type of bradyarrhythmic potential of risperidone seems to be reversible with dose-related conditions. Recent research works have reported that risperidone has electrophysiological properties like other class III antiarrhythmics, such as amiodarone and sotalol, which can cause sinus bradycardia and sinus pauses.[9] Some researchers suggest that risperidone leads to concentration-dependent block of the rapid component of the delayed rectifier K^+ current (I_K) in voltage-clamped canine ventricular myocytes.[9] Current research suggests that risperidone also causes class III antiarrhythmic properties, and it may result in QTc prolongation, particularly seen in patients with long QT syndrome and PVCs. Therefore, psychotic patients should be monitored with ECG during risperidone therapy.

Here, we describe a young man with schizophrenia without any prior cardiac history who developed symptomatic sinus bradycardia with pauses associated with palpitations even with a low dose of risperidone. The brady-arrhythmias resolved after discontinuation of risperidone. Symptomatic treatment, such as beta-blocker medications, should be initiated, and close ECG monitoring should be provided to the patient, implicating it in the pathogenesis of this patient's arrhythmia. Even though risperidone has a relatively low side effect profile with a low dosage, it is important to note that symptomatic cardiac side effects and death have been noted at moderate to high levels of risperidone, 6–24 mg/day.[10]

In our patient, we started aripiprazole because it is considered to be a relatively low-risk antipsychotic regarding cardiac risk. However, some case studies reported cardiac side effects, like atrial fibrillation after starting aripiprazole, but later the same medication was continued with an oral low dose of aripiprazole 5 mg/day and restarted long-acting aripiprazole on follow-up with a dose of 400 mg intramuscular injection every 4 weeks. He reported no recurrence of palpitation and other symptoms of arrhythmia and also showed improved psychosis with no further psychiatric hospitalization.[11]

Conclusion

We conclude that the brady-arrhythmic condition occurs due to risperidone therapy, but it is dose related and reversible. Even though it has safety efficacy, we believe that risperidone should be included in the progressively increasing category of drugs that induce brady-arrhythmias, so it should be monitored with the ECG during the therapy. Also, we conclude that the startoff and setoff of PVCs are temporally related to the startoff and setoff of the risperidone treatment.

Clinical Significance

It is very important for physicians to be vigilant about cardiac side effects, like PVC, and to do a regular close ECG monitoring of the patient during the treatment to prevent drug-induced arrhythmias.

Ethics Consent

The case report was approved by the Ethical Committee of Research and Studies Department, Directorate of Health Affairs, Taif (KSA).

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity.

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